Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting of April 7th, 1891.

DR. B. SACHS in the Chair.

THE PATHOLOGY OF LANDRY'S PARALYSIS.[1]

The case reported was that of an unmarried man, aged 45 years, free from any syphilitic or rheumatic taint, who was suddenly attacked by a paresis of the legs which, in the space of four days, became a complete paralysis, and then the muscles of the trunk, arms, and parts supplied by the bulbar nerves became paretic in the order named, and this paresis became a more and more decided paralysis until death from bulbar paralysis put an end to the futher extension of the process. The paralysis was a purely motor one, the sensory nerves not being involved, nor were the sphincters of the bladder and rectum involved, although the paralysis of the abdominal muscles made the expulsion of the contents of these viscera difficult or impossible. As the result of a careful microscopical examination of the organs by Dr. Van Gieson, the only lesions found were a slight cerebral and spinal meningitis of quite recent origin and a degeneration of a few of the fibres of the interior roots of the cauda equina.

After a careful consideration of those diseases of the nervous system most nearly resembling Landry's paralysis, the conclusion was reached that no evidence has been brought forward which justifies us in abandoning the term acute ascending or Landry's paralysis; for it has neither been shown that the numerous cases of the disease in which no lesion has been discovered depend on faulty methods of examination, nor has any case been reported which was clinically a typical case of Landry's paralysis in which after death characteristic lesions were found either in the central nervous organs or in the peripheral nerves. Acute ascending paralysis (defined so as to exclude all cases in which sensory symptoms are prominent,or in which well-marked bulbar symptoms are not present) must, therefore, be regarded as a clinical entity for which no corresponding lesion has as yet been discovered. That there is some change in the nervous system causing the severe symptoms cannot be doubted, but this change is probably of a chemical rather than of an anatomical character. From the many points of resemblance which acute ascending paralysis bears both to myelitis of the anterior horns and to multiple neuritis, this chemical change must effect either the motor cells of the spinal cord and medulla or the fibres springing from them ; and, although this chemical change is so great as to cause an entire arrest of the function of these cells or fibres, yet it leaves no trace in any altered character of cell or fibre, no more than does morphial or strychnial leave any trace in the structure of the nervous system of their fatal action.

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[1] Abstract of a paper on Acute Ascending (Landry's) Paralysis read by Dr. H. Hun before the New York Neurological Society, on April 7th 1891.

In regard to the nature of this supposed chemical poison we know nothing. The general tendency of the present day is to consider it to be a ptomaine, and indeed the acute course, the fact that it often follows an infectious disease, and that it is associated with an enlargement of the spleen, make it not improbable that Landry's paralysis is a germ disease. In further support of this view is the fact that Centanni and Eisenlohr have each found bacteria in the central nervous organs. In neither case was the bacteria cultivated. The case of Centanni was so imperfectly observed during life that the diagnosis even is doubtful, and in a second case, observed by Eisenlohr, no bacteria were found. In a number of cases bacteria was looked for as was done in the case reported, but was not found, so that the hypothesis that Landry's paralysis is due to bacterial agency, attractive as it seems, is far from being proven, and the pathology of the disease still remains to be discovered.

Dr. IRA VAN GIESON thought that Landry's paralysis was much more closely associated with cases of acute multiple neuritis, and acute poliomyelitis than was generally supposed. Landry, in 1859, had first accurately described several cases of ascending paralysis, although several observers had previously referred to the existence of such a disease. The speaker referred to several cases of acute multiple neuritis which had been reported in which the motor symptoms had predominated, and in which the whole course of the disease had been in close accord with that of Landry's disease. He also reported several known cases of acute poliomyelitis which resembled, very closely the disease in question. The clinical resemblance of these diseases was characteristic.

It was well known that multiple neuritis and poliomyelitis was usually caused by poisons of different kinds, such as the ptomaines resulting from the infectious diseases and from phthisis.

Etiologically, Landry's disease seemed to come under the same category as these two diseases. Before a definite conclusion could be arrived at, as to the true pathology of Landry's paralysis, a more thorough examination of all nerve trunks, with the branches and terminal filaments, would have to be made. Hitherto such observations have been unsatisfactory, because all the nerves could not be examined, for obvious reasons. For this consideration, the speaker thought that it should not be taken as conclusive, when it was stated that the peripheral nerves were not diseased in Landry's paralysis, as thorough examination might have proven the condition the very opposite. As to the hyaline thromboses found in the central nervous organs, such changes occurred in ptomaine poisoning, but were not confined to cases of Landry's disease, as it might be present in any case of poisoning.

Dr. LEONARD WEBER some six years ago had described a typical case of Landry's disease, in which the patient had entirely recovered in three months. In his case trauma, the shock of suddenly stopping a runaway horse, or the violent strain of pulling upon the lines, had caused the disease, as the attack had come on subsequent to this accident. He did not think that the trauma militated against the poison theory. He was satisfied that if a more thorough examination of the nerves were made in these cases, our idea of the pathology would become more definite.

Dr. M. A. STARR thought that it was a question whether the disease was of sufficient duration to cause neuritis and degeneration of the peripheral nerves, and whether such cases ever continued long enough for changes of this character to take place. It was certain that a myelitic process might cause a suspension of function without causing definitive microscopical changes. He had never seen a typical case of Landry's paralysis, but had seen two cases which presented many of its symptoms. One was manifestly a case of diptheritic paralysis, the other a case of beri-beri. He did not see how Dr. Hun could be satisfied that his case presented distinctive clinical symptoms. As for the case reported being like or bearing a resemblance to multiple neuritis, it certainly differed from any case the speaker had ever seen.